0842

10:00 h

IN VIVO PLATELET ACTIVITY IN YOUNG PATIENTS WITH CEREBRAL ISCHEMIA AND MITRAL-VALVE PROLAPSE.
R.E. Scharf, M. Hennerici, V. Bluschke and W. Schneider. Departments of Internal Medicine and Neurology, University of Düsseldorf, Düsseldorf, F.R.G.

Recent studies have demonstrated a sixfold higher incidence of mitral-valve prolapse (MVP) in young patients with cerebral ischemia compared to an age-matched control population. To examine whether there is a pathogenetic relationship between MVP and cerebral ischemia, we studied 47 patients (12 males, 35 females) under 45 (mean age 35.4 ± 6.6) years with transient ischemic attacks (TIA) and stroke who had failed to show a cause from extracranial Doppler examination, cranial computerized tomography and cerebral angiography. In vivo platelet activity was evaluated by measuring the ß-thromboglobulin (ß-TG) concentration in the platelet poor EDTA-theophylline-PGE_1-plasma using the Amersham RIA kit.

The ß-TG levels of the patients (54.9 ± 31.4, $\bar{x}$ ± SD ng/ml) were significant higher than those of an age- and sex-matched control group (n=40, 19.7 ± 6.4 ng/ml, $p < 0.001$) MVP was demonstrated in 28% of the patients (13 of 47) in contrast to 7.5% of the controls (3 of 40). The difference of the ß-TG levels of patients with MVP (n=13, 52.9 ± 25.5 ng/ml) and of patients without MVP (n=34, 55.7 ± 33.7 ng/ml) was not significant ($p < 0.40$). There was no correlation between duration or extent of the neurological deficit and the individual ß-TG level.

Our results confirm that the incidence of MVP is higher in young patients with cerebral ischemia of unknown cause than in asymptomatic young people. The significantly elevated plasma ß-TG concentrations in the patients' group may indicate an increased platelet activity in vivo. There was no significant difference between ß-TG levels of patients with and without MVP. Thus, MVP can not be the cause for the altered platelet activity.

0843

10:15 h

INCONSTANCY OF PGI_2 ADMINISTRATION IN PREVENTING ATTACKS OF PRINZMETAL ANGINA. S. Chierchia, R. De Caterina, F. Crea, W. Bernini, A. Distante, A. Maseri, A. L'Abbate. CNR Institute of Clinical Physiology. Pisa, Italy.

It has been proposed that vasospastic angina, eventually due to local defects of PGI_2 production, might benefit from PGI_2 administration. We therefore investigated the effects of PGI_2 in healthy volunteers and, then, in patients with frequent ischemic episodes (IE) of Prinzmetal angina, to determine 1. hemodynamic, antiplatelet and possible side effects of the drug and 2. its possible therapeutic usefulness in the management of IE. In 6 healthy volunteers PGI_2 was infused i.v. at doses of 2.5, 5, 10 and 20 ng/kg/min during consecutive periods of 30 min each. Heart rate (HR) and right atrial pressure were monitored continuously; cardiac output (thermodilution in 2 subjects, indirectly by a Doppler technique in all), arterial blood pressure (BP) and *in-vitro* platelet aggregability (PA) by ADP (Born), intermittently. In 2 subjects we also measured pulmonary arterial pressure and, in one, left ventricular pressure, during the infusion and in control conditions. PGI_2 was then infused in 6 pts with frequent IE at maximal well tolerable rate (6-26 ng/kg/min) for periods of 3 hours alternated with equal periods of placebo (P), continuosly recording 2 ECG leads to detect ST-T changes, and sampling blood for PA as before. In all healthy volunteers PGI_2, at the highest rates of infusion, decreased significantly ($p < .001$) both systolic BP (-10 ± 3%, mean ± SD) and diastolic BP (-19 ± 5%) increasing HR (+ 21 ± 5%); no significant changes were observed in the other hemodynamic parameters. The maximal decrease in PA was 58 ± 30% ($p < .001$). Skin flushing, restlessness and headache, sometimes observed at the highest doses, rapidly disappeared decreasing the infusion rate. In the 6 pts the same trend in BP, HR and PA was evident. 106 IE were observed. PGI_2 did not affect severity, duration and number of IE (44 during P, 62 during PGI_2 infusion). One of the pts, however, not clinically different from the others, showed a reduction at 10 ng/kg/min (6 IE during P, 2 during PGI_2) and a complete abolition in the 3 following periods at 20 ng/kg/min (4, 3, 5 IE during P vs. none during PGI_2).

We conclude that 1. PGI_2 can be safely administered to humans and 2. it may prevent IE is some vasospastic pts, but not in others. Different pathogenetic mechanisms are perhaps involved in apparently similar Prinzmetal anginas.

0844

10:30 h

THROMBOXANE B_2 AND BETATHROMBOGLOBULIN IN SYMPTOMATIC CORONARY ARTERY DISEASE. A.C. de Boer, A.G.G. Turpie, R. Butt, E. Genton, Department of Medicine, McMaster University, Hamilton, Ontario, Canada.

To investigate pattern and clinical value of platelet release and thromboxane synthesis in myocardial ischemia and necrosis, plasma (P) thromboxane B_2 (TXB_2) (normal 162 ± 28 pg/ml, mean ± SEM), P-betathromboglobulin (BTG) (24 ± 2 ng/ml) and urine (U) BTG (0.18 ± 0.04 ng/ml) were measured by radioimmunoassay in 98 patients admitted to the CCU with chest pain thought to be cardiac. Final diagnoses were 26 patients with non-cardiac pain (Gp I), 47 patients with angina (Gp II) and 25 patients with acute MI (Gp III). P-BTG and TXB_2 were measured at presentation and on day 1, 2 and 3, U-BTG on Day 1, 2 and 3. Gp I; P-BTG (26 ± 3; 20 ± 1; 24 ± 2; 22 ± 3), TXB_2 (170 ± 15; 178 ± 11; 195 ± 11; 162 ± 22), U-BTG (0.18 ± 0.04; 0.17 ± 0.05; 0.26 ± 0.08) were not different from normal. Gp II; P-BTG (38 ± 5; 35 ± 4; 38 ± 4; 39 ± 5) TXB_2 (525 ± 139; 358 ± 40; 356 ± 37; 330 ± 34) were different from Gp I on all days ($p<0.01$); U-BTG (0.38 ± 0.13; 0.14 ± 0.03; 0.14 ± 0.03) was not different from Gp I. Gp III; P-BTG (49 ± 12; 33 ± 3; 36 ± 5; 29 ± 3) and TXB_2 (237 ± 32; 289 ± 39; 285 ± 38; 340 ± 57) were elevated compared to Gp I ($p<0.01$), but not to Gp II, except for TXB_2 at presentation ($p<0.05$); U-BTG (0.49 ± 0.19; 0.40 ± 0.11; 0.16 ± 0.05) was not different from either Gp I or II. In Gp II, TXB_2 levels were higher ($p<0.01$) in 26 patients with recurrent episodes of ischemic pain (636 ± 238; 439 ± 56; 419 ± 51; 406 ± 42) compared to 21 stable patients (413 ± 149; 243 ± 46; 264 ± 48; 216 ± 39). Plasma BTG was normal in 61% of patients with established CAD, and the correlation with clinical course was poor. There was a weak correlation between P-BTG and TXB_2 ($r= 0.20$, $p<0.01$) and P and U-BTG ($r= 0.31$; $p<0.01$). These data indicate that platelet release and prostaglandin synthesis occur in a proportion of patients with coronary ischemic syndromes. TXB_2 is more often increased than BTG and occurs in most patients with recurrent ischemia. However, neither test has sufficient sensitivity to be of clinical value.

0845

10:45 h

PLASMA β-THROMBOGLOBULIN, PLATELET FACTOR 4 AND FIBRINOPEPTIDE A IN PATIENTS WITH CORONARY ARTERY DISEASE. A. Nichols, J. Owen, K.L. Kaplan, P.J. Cannon, H.L. Nossel, R. Sciacca. Department of Medicine, Columbia University College of Physicians & Surgeons, New York, NY, U.S.A.

To determine whether activation of platelets and coagulation is present in patients with coronary artery disease, plasma levels of platelet factor 4 (PF4), β-thromboglobulin (βTG), and fibrinopeptide A (FPA) were measured by radioimmunoassay in patients subjected to coronary angiography. The patients were divided into those with normal coronary angiograms (Group I, n = 14), those with coronary artery disease (> 70% narrowing) but no previous myocardial infarction (Group II, n = 32), and those with coronary artery disease and documented previous myocardial infarction (Group III, n = 36). The three groups did not differ in sex, incidence of hypertension or diabetes, serum cholesterol, HDL cholesterol, BUN or platelet count. Geometric mean values for the three groups were FPA: 0.77, 0.81 and 1.01 pmol/ml respectively, βTG: 22.7, 21.6 and 33.2 ng/ml respectively, and PF4: 5.7, 5.8 and 8.3 ng/ml respectively. When the data were tested by analysis of variance, significant elevations of βTG ($p< .01$) and PF4 ($p< .05$) were found in Group III but there were no other significant changes. When Group III was subdivided into patients with and without ventricular aneurysm, βTG and FPA levels were found to be higher in patients with aneurysm than without: βTG 45.9 vs. 30.3 ng/ml and FPA 1.64 vs. 0.88 pmol/ml ($p< .05$ for each). βTG levels were also higher in patients with congestive heart failure ($p< .01$) and showed an inverse correlation with left ventricular ejection fraction ($p< .05$) and a direct correlation with the extent of left ventricular asynergy ($p< .01$). In conclusion, elevations in βTG and PF4 were associated with previous infarction, not with coronary artery disease. These changes are thought to reflect platelet reaction with the damaged ventricular wall. Elevations in FPA were seen only in patients with ventricular aneurysm and may reflect mural thrombus within the aneurysm.

This document was downloaded for personal use only. Unauthorized distribution is strictly prohibited.